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Are Smarter Brains Running Faster? Heritability of Alpha Peak Frequency, IQ, and Their Interrelation

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It has often been proposed that faster central nervous system (CNS) processing amounts to a smarter brain. One way to index speed of CNS processing is through the assessment of brain oscillations via electroencephalogram (EEG) recordings. The dominant frequency (peak frequency) with which neuronal feedback loops in an adult human brain oscillate in a relaxed state is around 10 cycles/sec, but large individual differences exist in peak frequencies. Earlier studies have found high peak frequencies to be associated with higher intelligence. In the present study, data from 271 extended twin families (688 participants) were collected as part of a large, ongoing project on the genetics of adult brain function and cognition. IQ was assessed with the Dutch version of the Wechsler Adult Intelligence Scale (WAIS-IIIR), from which four dimensions were calculated (verbal comprehension, working memory, perceptual organization, and processing speed). Individual peak frequencies were picked according to the method described by Klimesch (1999) and averaged 9.9 Hz (*SD* 1.01). Structural equation modeling indicated that both peak frequency and the dimensions of IQ were highly heritable (range, 66% to 83%). A large part of the genetic variance in alpha peak frequency as well as in working memory and processing speed was due to nonadditive factors. There was no evidence of a genetic correlation between alpha peak frequency and any of the four WAIS dimensions: Smarter brains do not seem to run faster.

KEY WORDS: Neural speed; intelligence; twin study; electroencephalogram (EEG).

INTRODUCTION

The idea that faster central nervous system (CNS) processing may amount to a smarter brain has been proposed in earlier studies (e.g., Vernon, 1987) and has recently been supported by studies reporting positive relations between inspection time and IQ (Luciano *et al.*, 2001; Posthuma *et al.*, 2001). An alternative way to index speed of CNS processing is through the assessment of brain oscillations via electroencephalogram (EEG) recordings. Rhythmic activity measured with EEG scalp recordings derives from the summed synchronized synaptic activity of large populations of neurons (Steriade *et al.*, 1990). The dominant frequency (peak frequency) of this rhythmic activity in a relaxed state in adults is around 10 Hz, but large differences exist in individual peak frequencies (Lykken *et al.*, 1974; van Beijsterveldt and Boomsma, 1994; Klimesch, 1999; Osaka *et al.*, 1999). Previous studies have attempted to relate peak frequency to intelligence, arguing that a faster oscillating brain reflects rapid information processing, which in turn is associated with higher intelligence (e.g., Vogel and Broverman, 1964; Anokhin and Vogel, 1996; Osaka *et al.*, 1999), but this theory has long been debated (e.g., Ellingson, 1966; Ellingson and Lathrop, 1973; Vogel and Broverman, 1964).

In the past decade, experimental evidence has increased our understanding of the underlying physiological mechanisms responsible for brain oscillations, particularly in the alpha frequency range (Steriade *et al.*, 1990; Lopes da Silva, 1991). Generally, the alpha

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rhythm, as measured from the scalp, is defined to range between 8 to 13 Hz, occurs during wakefulness, and can be measured particularly over the occipital cortex. It appears when the eyes are closed and disappears when the eyes are opened (Berger, 1929). Alpha waves have been shown to be generated in thalamocortical feedback loops of excitatory and inhibitory nerve cells (Steriade *et al.,* 1990; Lopes da Silva, 1991). In the visual cortex, the alpha rhythm can also be generated by cortico-cortical networks involving layer V pyramidal neurons (Lopes da Silva and Storm van Leeuwen 1977; Steriade *et al.,* 1990). The specific alpha peak frequency of an individual is determined by the intrinsic membrane properties of the thalamic neurons projecting to the cortex (Steriade *et al.,* 1990).

Lebedev (1990, 1994) has proposed a functional role for the human alpha rhythm in stating that "cyclical oscillations in an alpha rhythm determine the capacity and speed of working memory. The higher the frequency the greater the capacity and the speed of memory" (Lebedev, 1994). In addition, Klimesch (1997) has argued that thalamo-cortical feedback loops oscillating within the alpha frequency range allow searching and identification of encoded information. He speculated that faster oscillating feedback loops would correspond to faster access to encoded information. These theories are supported by the results of some recent studies; Klimesch (1997) found that the alpha peak frequency of good working memory performers lies about 1 Hz higher than that of poor working memory performers. A study by Lehtovirta *et al.* (1996), comparing Alzheimer's patients with controls, found that alpha peak frequency of Alzheimer's patients was significantly lower than that of controls. This was explained in terms of cognitive slowing due to cholinergic deficits characteristic of Alzheimer's disease. It is also known that peak frequency tends to decrease with normal aging (Köpruner *et al.,* 1984). In summary, a theoretical neurophysiological framework as well as empirical evidence support the existence of a link between peak alpha frequency and (working) memory processes. Because working memory is a central component of intelligence (Daneman and Merikle, 1996; Engle *et al.,* 1999; Kyllonen and Christal, 1990; Necka, 1992), it seems reasonable to expect that alpha peak frequency is important to intelligence.

Metaphorically, the peak frequency of thalamocortical alpha activity can be hypothesized to determine the speed of encoding (and accessing) of information just like the processor speed of a microprocessor is determined by its basic clock cycle. High alpha peak frequency then is expected to be associated with high IQ. Only very few studies, however, have related alpha peak frequency successfully to measures of IQ. Anokhin and Vogel (1996) reported a correlation of 0.35 between alpha peak frequency and verbal abilities, but thus far this result has not been replicated (e.g., Jausovec and Jausovec, 2000). Also, no large study has provided a heritability estimate for alpha peak frequency (only a few small studies have appeared that reported twin correlations, e.g., Christian *et al.,* 1996). Although it has been speculated that the relation between alpha peak frequency and IQ is due to a genetic basis (e.g., Vogel, 2000), to our knowledge there have been no multivariate genetic studies reporting on the genetic correlation of alpha peak frequency with measures of IQ.

In the present study, we investigated whether and to what extent individual differences in alpha peak frequency can be attributed to genetic or environmental factors. In addition, the possible association between alpha peak frequency and each of the four dimensions of the WAIS-IIIR is decomposed into genetic and environmental components. An extended twin design (i.e., including families consisting of twins and additional siblings) is used to maximize statistical power to detect genetic and environmental influences (Posthuma and Boomsma, 2000).

METHODS

Subjects

Subjects were recruited from the Netherlands Twin Registry (Boomsma, 1998) as part of a large ongoing project on the genetics of cognition and adult brain function (Posthuma *et al.,* 2001; Wright *et al.,* 2001). Adult twins and their non-twin siblings were asked to participate in a 4.5-hour testing protocol. In one-half of the protocol, psychometric intelligence, inspection time, and reaction times were assessed; in the other half EEG activity was measured. The EEG registration included two noncognitive tasks that were analyzed for the present paper: 3 min resting EEG with eyes closed (EC) and 3 min resting EEG with eyes open (EO). The order of the two halves of the protocol was randomized across family members.

A total of 688 family members from 271 extended twin families participated in the study until December 2000. The complete sample consists of two age cohorts: a young adult cohort with a mean age of 26.2 years (*SD* 4.19) and an older adult cohort with a mean age of 50.4 years (*SD* 7.51). Participating families consisted of one

to eight siblings (including twins). On average, 2.5 subjects per family participated. In the young cohort, 171 males and 210 females participated, in the older cohort 135 and 172, respectively. The young cohort included 54 MZ pairs, 73 DZ pairs, 18 single twins, and 109 additional siblings. The older cohort included 48 MZ pairs, 58 DZ pairs, 15 single twins, and 80 additional siblings (for a detailed description of the sample characteristics see Posthuma *et al.,* 2001).

Intelligence Testing

IQ was measured with the Dutch adaptation of the WAISIII-R (WAIS-III, 1997). Dutch standardization norms for this version are currently being finalized, so it is not yet possible to report standard IQ scores. Individual scores for each subtest, except digit-symbol substitution, were calculated by weighting the observed score by the maximum possible score on that subtest times 100 (i.e., percentage correct on each subtest). For digit-symbol substitution the number of correct items per 60 sec was calculated. Nine subtests were administered. Subtest *information* measures general knowledge and information gathered from daily life. In subtest *similarities,* the subject is asked to describe in which aspect two verbally presented concepts are similar. In subtest *vocabulary,* the subject is asked to verbally describe the meaning of a specified term. Subtest *arithmetic* requires the subject to solve arithmetic questions within a certain time limit without paper and pencil. In subtest *letter-number sequencing,* the subject is asked to repeat a random sequence of up to eight numbers and letters and to put them in numerical and alphabetical order. In subtest *block design,* the subject needs to copy within a certain time limit a red and white pattern using red and white blocks. Subtest *matrix reasoning* requires the subject to decide which of five alternatives is most reasonably the missing part from a logical sequence. In subtest *picture completion,* the subject needs to state which essential part has been omitted from a given picture. In *digit-symbol substitution,* the subject needs to replace numbers with specified symbols as quickly and accurately as possible.

According to the WAIS guidelines (1997), the following four dimensions were calculated: Verbal Comprehension (VC; the mean percentage correct of subtests *information, similarities,* and *vocabulary*), Working Memory (WM; the mean percentage correct of subtests *arithmetic* and *letter-number sequencing*), Perceptual Organization (PORG; the mean percentage correct of subtests *block design, matrix reasoning,* and *picture completion*), and Processing Speed (PSPD; the number of correct items per 60 seconds of subtest *digit-symbol substitution*). The validity of these four dimensions was recently confirmed by a re-analysis of the WAIS manual data by Deary (2001).

EEG Administration

The EEG was recorded with 19 Ag/AgCl electrodes mounted in an electrocap. Signal registration was conducted using an AD amplifier developed by Twente Medical Systems (Enschede, The Netherlands). Signals were continuously represented online on a Nec multisync 17-in. computer screen using POLY 5.0 software (POLY, 1999) and stored for offline processing. Standard 10–20 positions were F7, F3, F1, Fz, F2, F4, F8, T7, C3, Cz, C4, T8, T7, P3, Pz, P4, T8, O1, and O2 (Jasper, 1958). Software-linked earlobes (A1 and A2) served as a reference. The vertical electrooculogram (EOG) was recorded bipolarly between two Ag/AgCl electrodes placed on the outer right canthus and 1 cm above the eyebrow of the right eye. The horizontal EOG was recorded bipolarly between two Ag/AgCl electrodes affixed 1 cm left from the left eye and 1 cm right from the right eye. An Ag/AgCl electrode placed on the forehead was used as a ground electrode. Impedances of all EEG electrodes were kept below 3 KΩ; impedances of the EOG electrodes below 10 kΩ. The EEG was amplified (0.05–30 Hz), digitized at 250 Hz and stored for offline processing. Dynamic regression analysis in the frequency domain (Brillinger, 1975) was used to minimize eye artifacts, especially rolling of the eyes in the eyes closed (EC) condition. During the EEG measurements, the subjects were seated in a comfortable reclining chair in a dimly-lit, sound-attenuated, electrically shielded room. A computer screen was placed 80 cm in front of them. Subjects were instructed to close their eyes, relax, and minimize movement during the 3-min EEG recording of the EC task. During the 3-min recording of the eyes open (EO) task subjects were instructed to fixate on the dot presented at the center of the computer screen and to avoid blinking.

Determination of Individual Alpha Peak Frequency (IAF)

Alpha peak picking is usually conducted on EEG recording of an EC condition by finding the maximum power within a certain frequency range. It is sometimes argued, however, that the "real" alpha peak occurs at that frequency which is most depressed by opening of

the eyes (e.g., Klimesch, 1999). In the present paper, the latter criterium was used to obtain accurate localization of the individual alpha peak frequency.

A power density spectrum was calculated by using a Fast Fourier Transform applied to 4-sec epochs of the 3-min recordings of each condition. This yielded 44 epochs (epoch 45 was not used for computational reasons) and a 0.25 Hz resolution in the power spectra. Because the occipital-parietal alpha rhythm can best be detected at occipital leads (depressed by opening of the eyes; Berger, 1929), O1 and O2 were chosen to calculate the power density spectra and the individual alpha peak frequencies (IAF). In the first 100 subjects, correlation of alpha peak frequency between O1 and O2 was found to be very near to 1, so one of the two occipital leads (O2) will be reported on only.

The peak frequency in the EC condition was determined as the highest peak in a window of 7 to 14 Hz in the EC power spectrum, irrespective of the shape of the spectrum. Visual inspection was conducted for peak frequencies occurring at the boundaries of the search window. Final localization of the correct IAF was based on an automated comparison between the peak frequency, as determined in the EC condition and the frequency at which alpha power was most depressed by opening of the eyes (i.e., finding the peak frequency in the spectrum obtained by subtracting the EO spectrum from the EC spectrum). If these two methods of peak detection yielded an identical peak frequency, this was taken as the IAF.

If the two methods yielded different peak frequencies (which occurred in 21% of the sample), the spectra were visually inspected in order to determine the real alpha peak frequency. For example, in cases where the EC spectra showed two peaks of approximately the same magnitude, that peak was taken at which alpha depression was highest.

Spectra with very low power (i.e., below 1.5 μV/Hz) and spectra with less than 44 epochs were removed from further analysis.

Statistical Analysis

Because the sample consisted of unbalanced pedigrees and had some missing data, models were fitted to the raw data instead of covariance matrices. This was accomplished by using the rectangular data file option in Mx (Neale, 1997). Four bivariate saturated models of IAF with each of the four WAIS dimensions were fitted in order to determine the fit of the four more restrictive bivariate variance decomposition models. The saturated models included a linear regression effect of age within each cohort and a deviation for males from the females within each cohort. The significance of these effects on the means were estimated in the saturated models. In addition, it was tested whether there was evidence for: (1) heterogeneity of variances across MZ twin pairs, DZ twin pairs, and siblings, across males and females, and across cohorts; (2) heterogeneity of correlations across MZM pairs and MZF pairs, and across DZM pairs, DZF pairs, DOS pairs, and sib-sib male/female pairings; (3) heterogeneity of DZ correlations and sib-sib correlations; (4) differences in means between MZ twin pairs, DZ twin pairs, and siblings; and (5) differences in means between age cohorts. The resulting most parsimonious saturated model is the model against which the bivariate variance decomposition models are tested.

In the bivariate variance decomposition models, the observed variance was decomposed in three of four possible latent sources of variance: additive genetic (A), shared environment (C) or non-additive (D), and nonshared environment (E) following Neale and Cardon (1992). For DZ twin pairs (and sib pairs if the saturated models indicated no difference in correlation between DZ twin pairs and sib pairs), similarity in shared environmental influences was fixed at 100%, similarity of additive genetic influences at 50%, similarity of nonadditive genetic influences at 25%, and no similarity in nonshared environmental influences. For MZ twin pairs, similarities of additive genetic, nonadditive genetic, and shared environmental influences were fixed at 100% and no similarity in nonshared environmental influences.

RESULTS

Of the complete sample of 688 subjects, 27 took the IQ test at home and did not participate in the EEG measurement session. Data from 12 subjects contained too many recording errors to be included in the peak picking procedure. In 18 cases, the IAF could not be picked due to very low-voltage power spectra. This left 631 subjects with an IAF. The mean IAF of the complete sample was 9.9 Hz (*SD* 1.01). Subjects with IQ test data, but without an IAF, were still included in the analyses.

Saturated Model Fitting Results and Descriptives

The saturated model fitting procedures indicated that for the individual alpha peak frequency and the four WAIS dimensions: (1) the variances were homogenous across sexes and across zygosities; (2) the MZF and

MZM correlations were homogenous; (3) the DZM, DZF, and DOS correlations were homogenous; and (4) the DZ correlations and sib pair correlations were homogenous. In addition, no differences in means were found between MZ twins, DZ twins, and sibs. However, the total variances and twin correlations across age cohorts were found to be statistically significant.

Table I shows the estimates of descriptive statistics in the most parsimonious saturated model, including significant effects on the means.

Males score higher than females in both age cohorts on all four WAIS dimensions except for Processing Speed. In contrast, males have a slower IAF, compared with females, in both the young and the older cohort. Please note that for IAF, the grand mean (10.03) represents the female mean (because no significant effects of age cohort and age within cohorts were found); the general mean, including females and males, was 9.9 Hz as previously stated.

All significant regression effects of age within the older cohort are negative, indicating that IAF and IQ scores decrease with age. In the young cohort, only the effect of age on Verbal Comprehension was statistically significant. The positive sign indicated an increasing score with age within the younger cohort.

The scores on the four WAIS dimensions in the older cohort are lower than the scores in the young cohort. From Table I it can be computed, for example, that for a male of average age (i.e., 26.18 years) in the young cohort the expected score for Verbal Comprehension is 51.39 + 0.47 * 26.18 + 3.86 = 67.55, whereas for a male of average age (50.39 years) in the older cohort the expected score is 51.39 + 22.50 − 0.28 * 50.39 + 5.92 = 65.70.

The phenotypic correlation between IAF and each of the four WAIS dimensions is calculated simultaneously with modeling the effects of age and sex on the observed scores. The correlations between IAF and each of the four WAIS dimensions were homogeneous over sex and ranged from −0.04 to 0.15. With one exception, none of the phenotypic correlations was statistically significant. The correlation of 0.15 between IAF and Working Memory in the older cohort was significant at an α of .05 ($\Delta\chi^2 = 4.96$, $\Delta df = 1$, $p = .026$), but was not significant at the Bonferroni corrected α of .006, correcting for multiple testing. The correlations were not dropped from the variance decomposition models, however, because a significant genetic correlation and a significant environmental correlation acting in opposing ways may result in a phenotypic correlation that is not different from zero.

The pattern of MZ and DZ correlations as estimated by maximum likelihood (ML) from the most parsimonious saturated model (Table II) suggests mainly genetic influences on IAF and the four WAIS dimensions. For IAF and Processing Speed in both cohorts, Working Memory in the young cohort, and Perceptual Organization in the older cohort, the MZ correlation is more than twice as high as the DZ correlation, suggesting nonadditive genetic influences. ADE models were fitted for these variables. For all other variables, ACE models were fitted.

Table I. Estimates of Descriptive Statistics of Individual Alpha Peak Frequency (IAF) and the Four WAIS Dimensions From the Final Saturated Model

	Correlation (IAF-IQ)		Effects on the mean					
	Young cohort	Older cohort	Grand mean	Deviation of older cohort	Regression weight of age in young cohort	Regression weight of age in older cohort	Deviation of males in young cohort	Deviation of males in older cohort
IAF	—	—	10.03	0	0	0	−0.18	−0.03
VC	0.06	−0.04	51.39	+22.5	+0.47	−0.28	+3.86	+5.92
WM	−0.04	0.15*	62.71	0	0	−0.15	+3.21	+8.56
PSPD	0.02	0.04	44.17	+10.26	0	−0.38	−4.69	0
PORG	−0.03	0.08	79.74	+12.07	0	−0.50	+4.03	+3.63

IAF = Individual alpha peak frequency.
VC = Verbal comprehension.
WM = Working memory.
PSPD = Processing speed.
PORG = Perceptual organization.
*Statistically significant at the 0.05 level.

Table II. MZ and DZ Correlations as Estimated by Maximum Likelihood From the Saturated Model in Two Different Age Cohort (see Table I for abbreviations)

		N pairs*	IAF	VC	WM	PSPD	PORG
Young	MZ	54 (47)	*0.73*	0.84	*0.70*	*0.62*	0.69
Cohort	DZ/sib pairs	283 (253)	*0.26*	0.46	*0.16*	*0.24*	0.34
Older	MZ	48 (44)	*0.83*	0.82	0.67	*0.70*	0.69
Cohort	DZ/sib pairs	242 (192)	*0.17*	0.45	0.34	*0.23*	0.25

*Number of pairs for IAF in brackets; sibpairs included all possible sib pairings within a family.
Italic: Variables for which an ADE model instead of an ACE model is fitted.

Variance Decomposition Model Fitting Results and Descriptives

Bivariate variance decomposition models of IAF and each of the four WAIS dimensions were fitted in order to determine the nature of the possible covariance between IAF and IQ. The statistical significance of the estimates in the full bivariate variance decomposition models was established by fitting nested models and comparing the fit statistic to the preceding model using the likelihood ratio χ^2 test. Results are presented in Table III. Equality of variances due to A, D, C, or E across cohorts was also tested and showed no differences in A, D or C, and E estimates for Verbal Comprehension, Processing Speed, and Perceptual Organization across cohorts.

Estimates from the full bivariate variance decomposition models are given in Table IV. Estimates in the most parsimonious variance decomposition models are given in Table V.

The observed phenotypic variance in IAF is mainly due to genetic variance. The genetic variance is decomposed into additive genetic variance (39%) and variance due to nonadditive generic influences (32%) in the young cohort. In the older cohort, only a very small part of the variance is ascribed to additive genetic variance ($<$1%) and the main genetic variance is due to nonadditive genetic variance (83%). Because models including nonadditive generic influences but excluding additive genetic influences are biologically implausible (Falconer and Mackay, 1996), the additive variance component is always retained in the model.

For the WAIS dimensions, except for Working Memory in the young cohort and Processing Speed in the both cohorts, a model which included an additive genetic component and a nonshared environmental component best fit the data. For Working Memory in the young cohort and Processing Speed in the both cohorts, however, the nonadditive genetic component could not be dropped from the model without significantly worsening the fit. The variance due to nonadditive genetic influences in Working Memory in the young cohort was large (71%). Estimates of the genetic and nonshared environmental variance components of Processing Speed were homogeneous across cohorts. Thirty-two percent of the total variance was due to additive genetic influences, 34% to nonadditive genetic influences, and 34% to nonshared environmental influences. Also, for Perceptual Organization and Verbal Comprehension, no difference in variance components estimates was found between cohorts; 68% and 83%, respectively, was due to additive genetic influences, and 32% and 17%, respectively, to nonshared environmental influences.

All common pathways from the bivariate variance decomposition models could be dropped, except the common nonshared environmental factor between IAF and Verbal Comprehension in the young cohort and the common nonshared environmental factor between IAF and Working Memory in both cohorts. The corresponding nonshared environmental correlation was 0.31 and the corresponding phenotypic correlation 0.07 for IAF and Verbal Comprehension. For IAF and Working Memory, the nonshared environmental correlation was 0.17 and the corresponding phenotypic correlation was 0.05 in the young cohort and 0.04 in the older cohort.

In conclusion, although a high heritability for IAF and all four WAIS dimensions was found, no genetic correlation between IAF and any of the four measures emerged.

Post-hoc Investigation

Because it is known that head size and alpha peak frequency tend to correlate negatively (Nunez *et al.*, 1978), whereas head size and IQ correlate positively (e.g., Jensen, 1994), we conducted *ad hoc* tests in SPSS to determine whether mediating effects of head size

Table III. Fit Statistics of Nested Bivariate Variance Decomposition Models of Individual Alpha Peak Frequency (IAF) and Each of the Four WAIS Dimensions

		χ^2*	*df**	$\Delta\chi^2$**	Δdf**
IAF	*Verbal Comprehension*				
ADE young, ADE older	ACE young ACE older with A-/E-correlation young and older	8.70#	2	—	—
ADE young, ADE older	ACE young = ACE older with A-/E-correlation young and older	8.83	5	0.13	3
ADE young, ADE older	ACE young = AE older with A-/E-correlation young and older	9.69	6	0.86	1
ADE young, ADE older	AE young = AE older	19.20#	10	9.51#	4
ADE young, ADE older	**AE young = AE older, with E-correlation young**	**12.20**	**9**	**2.51**	**3**
IAF	*Working Memory*				
ADE young, ADE older	ADE young, ACE older with A-/D-/E-correlations young, A-/E-correlations older	1.62	1		
ADE young, ADE older	AE young, ACE older with A-/D-/E-correlations young, A-/E-correlations older	9.17#	3	7.55	2#
ADE young, ADE older	ADE young, AE older with A-/D-/E-correlations young, A-/E-correlations older	1.62	2	0.00	1
ADE young, ADE older	ADE young, AE older	25.40#	7	23.78	6#
ADE young, ADE older	ADE young, AE older with A-/D-/E-correlations young, A-/E-correlations older and E-correlation young = E-correlation older	1.687	3	0.07	1
ADE young, ADE older	ADE young, AE older with A-/D-correlations young, A-correlation older	11.36	4	9.67#	1
ADE young, ADE older	ADE young, AE older with A-correlation young, A-correlation older with E-correlation young = E-correlation older	2.25	4	0.56	1
ADE young, ADE older	ADE young, AE older with A-correlation older with E-correlation young = E-correlation older	4.17	5	1.92	1
ADE young, ADE older	**ADE young, AE older with E-correlation young = E-correlation older**	**7.05**	**6**	**2.89**	**1**
IAF	*Processing Speed*				
ADE young, ADE older	ADE young, ADE older with A-/D-/E-correlations	0.48	0		
ADE young, ADE older	ADE (young = older) with A-/D-/E-correlations	1.18	3	0.70	3
ADE young, ADE older	**ADE young = ADE older, no correlation**	**6.55**	**9**	**5.38**	**6**
IAF	*Perceptual Organization*				
ADE young, ADE older	ACE young, ADE older with A-/E-correlations young, A-/D-/E-correlations older	0.81	1		
ADE young, ADE older	AE young, AE older with A-/E-correlations young, A-/E-correlations older	3.03	4	2.22	3
ADE young, ADE older	AE young = older with A-/E-correlations young=older	3.09	6	0.06	2
ADE young, ADE older	**AE young = AE older, no correlation**	**4.66**	**10**	**1.57**	**4**

*Against saturated model.

**Against less restrictive model.

Bold indicates most parsimonious model; # fits significantly worse than saturated/less restrictive model at $\alpha < .05$.

Note: In this table results are shown for dropping more than one effect at a time. The significance of each effect, however, was also tested by itself. This means, for example, that when dropping 5 effects at a time resulted in a χ^2 of 8.83, it was tested whether dropping each effect of these 5 by itself exceeded the critical χ^2-value of 3.84 for a 1 *df*-test.

Table IVa. Variance Decomposition Estimates (95% CI) in the Full Bivariate Models

		%A	%D	%C	%E
Young	IAF	40 (0−74)	31 (1−78)	—	28 (18−42)
	VC	74 (56−87)	—	9 (0−26)	17 (12−23)
	WM	11 (0−57)	59 (11−78)	—	30 (21−45)
	PSPD	33 (2−65)	33 (1−66)	—	35 (26−46)
	PORG	69 (34−79)	—	0 (0−26)	31 (21−46)
Older	IAF	1 (0−54)	82 (28−89)	—	17 (11−28)
	VC	74 (56−87)	—	9 (0−26)	17 (12−23)
	WM	67 (28−78)	—	0 (0−27)	33 (22−51)
	PSPD	33 (2−65)	33 (1−66)	—	35 (26−46)
	PORG	37 (0−76)	32 (0−77)	—	31 (20−50)

may have blurred a positive relation between IAF and IQ. Head circumference was measured with a measuring tape. In the complete sample, the correlation between IAF and head size was −0.12 ($p = 0.003$). This negative correlation was mainly due to a negative correlation between IAF and head size in females from the young cohort (−0.17, $p = 0.016$).

The correlations in the complete sample between head size and each of the four WAIS dimensions were all significant ($p < 0.001$), except for Processing Speed. For Perceptual Organization, Verbal Comprehension, and Working Memory the correlations were 0.15, 0.20, and 0.23, respectively.

When, in the complete sample, the correlation between IAF and each of the four WAIS dimensions was corrected for the effects of head size (in addition for correcting for the effects of age), still no correlation between IAF and any of the WAIS-dimensions was found. Also, when the dataset was divided into the four groups of young females, young males, older females, and older males, no correlation between IAF and any of the WAIS dimensions was observed after correction for head size, although for the older males the correlations of IAF and verbal comprehension and IAF with working memory were almost significant (0.17 with $p = 0.063$ and 0.16 with $p = 0.081$, respectively). In conclusion, mediating effects of head size did not explain the absence of a relation between IAF and IQ.

DISCUSSION

The present study, which includes a large representative sample of healthy Dutch adults, is the first large study to report heritability estimates of alpha peak frequency. It is also the first study to investigate the genetic and/or environmental correlation between alpha peak frequency and IQ. No significant correlation between alpha peak frequency and IQ at either the genetic, environmental, or phenotypic level was found, with the exception of a small correlation of peak frequency with Working Memory and Verbal Comprehension in the older cohort. These correlations were

Table IVb. Estimates (95% CI) of Genetic and Environmental Correlations in the Full Bivariate Models

		A correlation with IAF	D correlation with IAF	E correlation with IAF
Young	IAF	—	—	—
	VC	0.05 (−1.00–1.00)	—	0.30 (0.04–0.51)
	WM	−1.00 (−1.00–1.00)	0.25 (−0.95–1.00)	0.18 (−0.10–0.43)
	PSPD	−0.98 (−1.00–0.24)	0.83 (−0.28–1.00)	0.09 (−0.16–0.33)
	PORG	−0.08 (−1.00–1.00)	—	0.03 (−0.21–0.27)
Older	IAF	—	—	—
	VC	0.05 (−1.00–1.00)	—	0.08 (−0.20–0.35)
	WM	1.00 (0.22–1.00)	—	0.12 (−0.18–0.40)
	PSPD	1.00 (−0.84–1.00)	−0.29 (−1.00–0.61)	0.10 (−0.93–0.39)
	PORG	1.00 (−1.00–1.00)	−0.33 (−1.00–1.00)	0.06 (−0.23–0.35)

Table V. Estimates (95% CI) in the Most Parsimonious Bivariate Variance Decomposition Models

		%A	%D	%E	E correlation with IAF
Young	IAF	39 (0–74)	32 (1–80)	29 (18–44)	—
	VC	83 (78–87)	—	17 (12–22)	0.31 (0.04–0.50)
	WM	0 (0–53)	71 (16–80)	29 (20–43)	0.17 (0.01–0.33)
	PSPD	32 (1–69)	34 (1–72)	34 (26–46)	—
	PORG	68 (57–76)	—	32 (24–43)	—
Older	IAF	0 (0–62)	83 (28–89)	17 (11–28)	—
	VC	83 (78–87)	—	17 (12–22)	—
	WM	67 (50–78)	—	33 (22–50)	0.17 (0.01–0.33)
	PSPD	32 (1–69)	34 (1–72)	34 (26–46)	—
	PORG	68 (57–76)	—	32 (24–43)	—

Note: In Tables IVa and IVb, estimates for IAF are taken from the bivariate model IAF with VC, which was representative of all four bivariate models. This causes slightly different parameter estimates for IAF in Table IVb compared with Table IVa.

completely mediated by a common nonshared environmental factor. Because they did not survive the Bonferroni correction for multiple testing, however, these correlations should be regarded with caution.

The absence of a genetic correlation between alpha peak frequency and any of the four WAIS dimensions in this study suggests that genetic differences among individuals in the speed with which the thalamo-cortical feedback loops within the brain oscillate do not contribute to differences among individuals in IQ. This result is at odds with findings in previous studies. Studies in subjects with mental retardation (see Ellingson, 1966; Vogel and Broverman, 1964 for a review of the early studies) or Alzheimer's disease (Lehtovirta *et al.*, 1996; Klimesch, 1997) most clearly show that when the brain is not functioning optimally, both alpha peak and IQ are depressed. In addition, however, a significant link between alpha peak frequency and IQ has been found in populations with a normal IQ range (e.g., Klimesch *et al.*, 1996; Klimesch, 1999; Köpruner *et al.*, 1984; Lebedev, 1994; Osaka *et al.*, 1999; Anokhin and Vogel, 1996). Compared with these previous studies, our study differs mainly in the operationalization of IQ; in the present study, the correlation between alpha peak frequency and WAIS dimensions was investigated. Most previous studies did not use the WAIS to measure IQ (e.g., Klimesch *et al.*, 1996; Klimesch, 1997, 1999; Lehtovirta *et al.*, 1996; Köpruner *et al.*, 1984; Lebedev, 1994; Osaka *et al.*, 1999). Anokhin and Vogel (1996) did use a measure similar to the WAIS to tap general IQ, spatial IQ, and arithmetic abilities and found no significant correlation with any of these and alpha peak frequency. However, in the same study, a significant correlation of 0.35 between alpha peak and verbal abilities, as measured by the Amthauer's Intelligence Structure Test and Horn's Leistungsprüfsystem test, was reported, suggesting that alpha peak frequency may correlate with very specific mental abilities and may not be related to general IQ. Klimesch (1997), who repeatedly linked high alpha peak frequency to good memory performance, used several tests other than the WAIS to tap both working memory and memory: a Sternberg test, a verbal recognition test, an experimental learning test, and an incidental learning test.

A possible explanation for the absence of a correlation between alpha peak frequency and scores on the WAIS-dimensions could be that neural speed *per se* does not play a prominent role in general IQ. Rather, the degree of connectivity between areas or the total gray and white matter (brain volume) may be of greater importance. In other words, efficient interconnectivity of the brain could result in high processing speed without the need of fast oscillating thalamo-cortical feedback loops. Studies relating coherence (a measure of connectivity of the brain) to IQ have indeed reported a relation between efficient connectivity and measures of intelligence (e.g., Jausovec and Jausovec, 2000; Anokhin *et al.*, 1999).

Alpha peak frequency was shown to be highly heritable: In the young adult cohort, 71% of the total variance could be ascribed to genetic variance; in the older cohort this was 83%. These estimates of heritability are among the largest heritabilities reported for a quantitative trait (Plomin and DeFries, 1990). A large part of the genetic variance was estimated to be caused by nonadditive genetic variance; 32% in the young cohort and

83% in the older cohort. Nonadditive genetic variation can be either dominance variation or epistatic variation or both. Dominance variation of a trait refers to the variation due to the interaction effect of the two alleles that define the genotype at a locus. Dominance is distinct from the interaction that may occur between genotypes at separate loci (i.e., epistasis). However, these sources of variance are confounded in the classical twin study (i.e., including only MZ twin pairs and DZ twin pairs/sib pairs) as in most non-experimental genetic studies.

A large estimate of nonadditive influences and a near-zero estimate of additive genetic influences was found for alpha peak frequency in the older cohort. However, the confidence intervals around the estimates of nonadditive genetic variance and additive genetic variance are very broad and highly overlapping, indicating the difficulty in the separate detection of these two influences. The real additive variation and real dominance variation could be anywhere between 1% and 74%, or 1% and 80%, respectively. In the classic twin design, estimates of nonadditive genetic influences and additive genetic influences are highly negatively correlated (-0.9), resulting in stable broad heritability estimates, but large fluctuations in the estimates of these two influences (Eaves, 1972). Including subjects of many different genetic relationships (e.g., MZ twin pairs. DZ twin pairs, half siblings, parent-offspring) will increase the reliability to separate additive from nonadditive genetic influences.

An alternative explanation for the large estimate of nonadditive genetic influences may be that the observed DZ correlation was slightly lower than the true DZ correlation. This bias may occur when twins are sampled from a truncated distribution, which may lead to a slightly misrepresented sample. Martin and Wilson (1982) showed that this selection reduces the correlation between twin pairs and has a proportionally larger effect on lower correlations as compared to higher correlations. This, in turn, may easily result in the estimation of huge nonadditive genetic effects and zero additive genetic effects. For example, when the true MZ correlation is 0.8 and the true DZ correlation is 0.3, the corresponding true percentages of the total variation explained by additive and nonadditive genetic influences are 40% and 40%, respectively. However, if the observed correlations are 0.8 for MZs and 0.2 for DZs, the percentage of observed variation explained by additive genetic influences is estimated to be zero and the percentage of variation explained by nonadditive influences is estimated to be 80% (the percentage of variation explained by nonadditive genetic influences can quickly be obtained by calculating ($2 \times$ MZ-correlation minus $4 \times$ DZ-correlation) $\times$ 100).

Although the magnitude of the nonadditive genetic influences on alpha peak frequency is likely to be overestimated, other studies support our results in suggesting the presence of nonadditive genetic effects in at least some loci. Lykken *et al.* (1982) reported an MZ correlation of 0.81 and a DZ correlation of -0.15. Although Lykken *et al.* (1982) did not test the departure from the additive model nor estimate the proportion of nonadditive influences, their results were explained in terms of dominance, epistasis, and gene-environment interactions. Christian *et al.* (1996) did estimate the influence of both dominance variance and variance due to epistasis. They found no evidence of additive genetic variance on alpha peak frequency, but the dominance variance and the epistatic variance were estimated at 21% and 18%, respectively.

An alternative explanation for MZ correlations to be more than two-fold the DZ correlation is a specific MZ environment (Wyatt, 1993). Because the twin correlations on EEG parameters for MZs reared together and MZs reared apart are similar (van Beijsterveldt and Boomsma, 1994), this specific MZ environment can only reasonably be sought in a more similar prenatal environment for MZs compared with DZs. It is known that a dysfunctional prenatal environment may result in dysfunctional neuropsychological functioning, as measured by EEG (Scher, 1997a, 1997b). When, for example, MZs are exposed to a specific prenatal environment that causes them to have more similar alpha peak frequencies later in life, the MZ correlation will be inflated compared with the DZ correlation and will falsely result in an estimation of nonadditive genetic influences (Christian *et al.*, 1975). However, such an effect will also be present in different mean alpha peak frequencies. In the present study, no mean differences were found between MZs, DZs and sibs. In addition, when MZs are under the influence of an additional source of variance (i.e., their specific prenatal environment) the result will be a greater total variance for MZ twin pairs compared with DZ twin pairs and sibs. Again, we found no evidence for a difference in variance as a function of zygosity. The nonadditive genetic influences in alpha peak frequency thus appear to be genuine nonadditive genetic influences.

While addressing its primary question, this study uncovered a number of noteworthy findings on the genetic architecture of the IQ dimensions. As expected, differences among individuals in the four WAIS dimensions could be attributed to genetic factors and non-

shared environmental factors, but not to shared environmental factors. The absence of shared environmental influences on specific cognitive abilities measures in adults is consistent with reports from other studies (Plomin *et al.,* 1994a, 1994b). On average 70% of the total interindividual variance was accounted for by additive genetic factors for Verbal Comprehension in both cohorts, Perceptual Organization in both cohorts, and Working memory in the older cohort. For Perceptual Organization in the older cohort, the full variance decomposition model estimated a moderate amount of nonadditive genetic variance that did not reach significance. However, for Processing Speed in both cohorts and Working Memory in the young cohort, the nonadditive genetic variance was significantly different from zero and explained 34% and 71% of the total variance, respectively.

The presence of nonadditive genetic variance in specific cognitive abilities or IQ in general is not often explicitly tested for, presumably because only very large samples have enough statistical power to detect it (Martin *et al.,* 1978). The large amount of variance due to nonadditive genetic sources in combination with the use of an extended twin design (Posthuma and Boomsma, 2000) gave enough power to detect nonadditive genetic variance in the present study, although the detection was not very accurate as indexed by the broad confidence intervals. As discussed earlier, very large sample sizes and information from many different genetic relationships between subjects are needed to separate additive genetic influences from nonadditive genetic influences reliably.

The presence of nonadditive genetic influences on cognitive abilities in the present study complies with the early work of Jinks and Fulker (1970), who reanalyzed the IQ data of Burt and Howard (1956) and concluded that ". . . dominant gene action for IQ almost certainly exists." Reported MZ and DZ correlations from some recent IQ studies also suggest the influence of nonadditive genetic variance. For example, Plomin *et al.* (1994b) reported an MZ correlation of 0.60 and a DZ correlation of 0.08 for the WAIS digit span subtest in the SATSA sample of 67-year-old subjects. In addition, for several other subtests the reported MZ and DZ correlations were suggestive of nonadditive genetic variance. However, nonadditive genetic variance was not included in the analyses.

Fulker and Eysenck (1979) noted that for ". . . many genes influencing IQ there is a marked degree of dominance." Evidence from human inbreeding studies, they argued, clearly indicate the presence of recessive alleles for low IQ and dominant alleles for high IQ. Normally, offspring show a regression to the population mean; i.e., the children of parents who are of lower-than-average IQ tend to be of average IQ as well as the children of parents of higher-than-average IQ. If the IQ of children of blood-related parents tends to be lower than that of children from unrelated parents, then there is evidence of recessive alleles influencing low IQ. This is exactly what was observed in an Israeli study by Bashi (1977); controlling for socioeconomic status, children born to biologically related parents were of lower IQ than children born to unrelated parents. In fact, children born to double first cousins showed a larger adverse effect than children born to first cousins. Another study by Seemanova (1971) found that the IQ of 161 children born from incestuous relationships was severely depressed. In contrast, the IQ of 95 children born to the same mothers but from a different relationship was completely normal. These findings clearly suggest the existence of recessive alleles decreasing IQ and, more generally, of nonadditive genetic variation in IQ.

In conclusion, this study, which included 688 healthy Dutch adult family members, showed that both alpha peak frequency and specific cognitive abilities, as measured with the WAIS, were highly heritable. Possibly as a consequence of the large sample size and the power added by the extended twin design, significant evidence was obtained for nonadditive genetic influences on IQ and on alpha peak frequency. No association between alpha peak frequency and WAIS-IQ at either the genetic, environmental, or phenotypic level was found.

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